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**Drug Assay Method.**—An attempt to devise a rapid and economical method of assaying the tuberculostatic property of compounds resulted in the following procedure: serum of animals that had received the drug to be tested was incorporated in a liquid medium which was then inoculated with tubercle bacilli of human type. The presence or absence of an inhibitory action of a compound on the growth of the culture could be observed and its effect *in vivo* could be anticipated. This suggests a method for overcoming the usual discrepancy between the results of *in vitro* and *in vivo* testing of substances for their activity against the tubercle bacillus.—*Procedure for Distinguishing Drugs Effective against the Tubercle Bacillus by a Combined in vivo and in vitro Method, E. F. White & A. G. Karlson, Proc. Staff Meet., Mayo Clin., January 21, 1948, 23: 52.*—(*P. Q. Edwards*)

**Virulence of Tubercle Bacilli.**—It is almost unanimously agreed that the newly formed tubercle bacilli growing on artificial culture media do not all become acid fast until about the fifth or sixth day of their development. What degree of virulence the germs have in this early period of their existence is not well known. It has, however, been shown recently that during the first five days of their growth in Sauton's synthetic media, tubercle bacilli are definitely less virulent than in older cultures. The author followed the development of pathogenicity of bovine tubercle bacilli developing in the upper levels of a liquid culture medium such as the egg medium of Besredka, following the inoculation of the lower portion with tubercle bacilli introduced through a glass tube. The germs developing at the upper levels of the medium would all be young, those introduced to start the culture remaining lower down. Samples were removed from the middle level after two, three, four, five, six, and twenty-one days. No acid-fast bacilli were found until after the second day. It was consequently assumed that samples removed on the third day would contain young bacilli. Dilutions of the samples in physiologic salt solution were made in various strengths (1:5, 1:10, 1:100, etc.). One-tenth cc. of each was planted on Lowenstein's medium, and 1 cc. was injected subcutaneously into guinea pigs and intravenously into rabbits. The number of bacilli in one cc. of each dilution was determined by counting the number of colonies that developed on the Lowenstein's medium. The lesions which developed in the animals inoculated with a corresponding number of germs made possible a comparison of the virulence of young germs (three to four days' growth) and older ones (six to twenty-one days). It was found that the difference was slight if only a small number of bacteria (less than 100) was employed. To secure more definite proof it was necessary to inoculate at least 100 to 500 bacilli; better results were secured when more than 500 were used. The guinea pigs and rabbits inoculated with bacilli removed from the medium on the third or fourth day of their development were examined postmortem, six weeks to three months after their inoculation. Very few tubercles were found anywhere, and the organs were apparently normal. On the other hand, animals inoculated with corresponding quantities of older bacilli (six days) and sacri-

ficed after the same period of waiting, had many tubercles in various organs. Their lesions were much more serious than those caused by bacilli three or four days old but not as extensive as those caused by bacilli twenty-one days old. In the latter case the tubercles were often confluent and generalized through all the organs. The author concludes that young bacilli in the first week of their growth have less virulence than those three weeks old. The pathogenicity which then characterizes them begins about the sixth day. The period during which the germs have only slight virulence corresponds with that during which many of them have not yet become acid-fast. These experiments are similar to those made by other workers, including P. Hauduroy (Le probleme des tuberculoses atypiques, Lausanne, 1946) who believes that the tubercle bacillus goes through an evolutionary cycle and that old and degenerating bacilli may also lack acid-fastness.—*Etude de pouvoir pathogène des bacilles tuberculeux, L. Nègre, Selection, April, 1947, 1: 208.—(A. T. Laird)*

**Experimental Tuberculosis in the Syrian Hamster.**—On account of the expense of feeding guinea pigs and rabbits, their slow rate of reproduction and the care required to rear them, the author, who is laboratory chief of the Laval Hospital in Quebec, has experimented with the golden or Syrian hamster (Cricetus Auratus). This is a small rodent of the Murides family, somewhat smaller than a Norway rat. The animal is about 30 cm. in length, has a short tail and is provided with two capacious pouches beginning one in each cheek and extending to its shoulders, which it stuffs with grain to store for the winter. It lives in underground passages where it hibernates. It is easy to care for, neat and clean, and in captivity it is omnivorous. Other advantages which it presents for experimental work is its short period of gestation, sixteen days, and the number of its offspring, from 5 to 11 per litter. These periods occur in rapid succession until hibernation which, however, lasts for about six months. It is relatively gentle if carefully handled. The animal has been used to some extent in the study of tropical diseases and parasitic infections. The author wished to determine its suitability for use in studying tuberculosis. The three animals of a first group, weighing about 110 grams each, were given subcutaneous injections of 0.1 mg. of a strain of human type bacilli of average virulence. Infection proceded very slowly and generalized tuberculosis developed only after three months. A second group of five was inoculated with 0.5 mg. of a virulent bacillus which had been used previously in experiments with guinea pigs and rabbits. They were killed after intervals varying from fifteen days to four months. Tuberculosis developed slowly in this group also. The third group of five animals was inoculated with 0.5 mg. of a strain of bovine type tubercle bacilli. These animals showed a more rapid development of tuberculosis. The author concludes that the Syrian hamster is not as useful for animal inoculation with tuberculosis as the guinea pig, but that it might be employed with advantage in differentiating the human and bovine types of tuberculosis. His findings confirm those of previous observers.—*Tuberculose experimentale du hamster dore, M. Giroux, Laval med., October, 1947, 12: 863.—(A. T. Laird)*

**Serum Albumin and Tween Medium.**—It has been previously reported that although the presence of commercial Tween 80 (an ester of oleic acid) facilitates submerged growth of tubercle bacilli in liquid media, initiation of growth with small inocula requires the addition of serum albumin. It was also found that the chief inhibitor of growth against which the albumin protects the tubercle bacilli is some unesterified oleic acid present in the commercial Tween 80 and further released from it during incubation. In the present paper the bacteriostatic effect of Tween 80 and the growth promoting effects, if any, of other proteins are reported. Also the effect of albumin on surface growth was studied. Direct proof that the Tween 80 molecule itself is not bacteriostatic was obtained by the preparation of material essentially free from fatty acid. Tween

80, freed of contaminating fatty acid, not only permitted growth of small inocula, but protected against small amounts of added oleic acid. It is unlikely that serum albumin in itself offers any nutriment for the tubercle bacillus. It is a protective bacterial growth factor; by binding traces of fatty acid in the medium it permits the unmolested growth of the smallest possible inoculum of tubercle bacilli. It is likely that the growth promoting effect of serum albumin on surface growth, when added to Long's synthetic medium (which contains no Tween), is due to its ability to bind the traces of fatty acid often present. For this purpose undenatured albumin is required. Crystalline beta-lactoglobulin has a smaller capacity, and a number of other proteins have no perceptible capacity to bind oleic acid.—*The Binding of Fatty Acids by Serum Albumin, a Protective Growth Factor in Bacteriological Media, B. D. Davis & R. J. Dubos, J. Exper. Med., September, 1947, 86: 215.*—(*J. S. Woolley*)

**Effect of Lipase on Bacterial Growth.**—Serum albumin is essential for the initiation of growth by small inocula of tubercle bacilli in a liquid medium containing "Tween 80" which promotes submerged growth of these organisms. The albumin acts as a protective rather than a nutritive growth factor binding traces of unesterified fatty acid which inhibit growth of small inocula. The hydrolysis of "Tween 80" by lipase in commercial albumin releases free fatty acid in bacteriostatic quantities exceeding the binding capacities of the albumin. Elimination of lipase permits the cultivation of small inocula such as 2 or 3 bacteria. Lipolysis was also found to account for the bacteriostatic effect of several materials of biological origin (serum, bacterial culture filtrates) observed only in media containing "Tween 80." The effect can be eliminated by the destruction of the lipase by heating at 56 C, by the use of commercial crystalline bovine serum albumin which is not perceptibly contaminated by lipase or by the addition to the medium of 0.01 per cent NaF, which inhibits lipase but is not bacteriostatic to the tubercle bacillus. The effect can also be overcome by the use of a high concentration of albumin. Lipolytic activity also accounts for the bacteriostatic effect in this medium of horse serum and of a culture filtrate of *M. phlei*. This experience emphasizes the importance of guarding against lipolytic effects when materials of biological origin are introduced into a Tween-containing medium for diagnostic work (*e.g.*, blood culture) or chemotherapeutic studies (*e.g.*, impure antibiotics). The use of "Tween 80" and tubercle bacilli provides an exceedingly sensitive bioassay for lipase. Although the bacteriological work was confined to human type tubercle bacilli, the principles evolved are applicable to the cultivation, in the presence of esters, of any bacteria that are sensitive to free fatty acid.—*The Inhibitory Effect of Lipase on Bacterial Growth in Media Containing Fatty Acid Esters, B. D. Davis & R. J. Dubos, J. Bact., January, 1948, 55: 11.*—(*F. G. Petrik*)

**Nature of Acid-fastness.**—Evidence is presented which suggests that the property of acid-fastness of the tubercle bacillus is dependent upon the permeability of the cytoplasmic membrane. When the bacillus is stained by the Ziehl-Neelsen method the dye gains entrance into the cell through this membrane where a small portion of the dye is firmly absorbed by the cytoplasm and the remainder is free. The free dye can be removed by immersion in boiling neutral 50 per cent alcohol or boiling water without altering the acid-fast property. In the presence of "Tween 80" the dye is removed in thirty to sixty seconds as compared with five to seven minutes in the absence of Tween and more rapidly in boiling alcohol than in boiling water. The free dye can be precipitated to form beads and these bodies are often greater in diameter than the width of the cell. The location of beads cannot be predicted before formation, nor can evidence of their previous location be found after their dispersal. When reformed they may be found in their original location, but just as often they occupy new positions in the cell. The granular form may be stained to show

beading but it was noted that the dye accumulates and is dispersed only in the granular portions and not in the unstained interspaces. The fact that tubercle bacilli are rendered non-acid-fast by trauma, autolysis, or the use of acid is satisfied by the proposed theory: the action has been so severe that the cytoplasmic membrane no longer controls permeability with relation to the dye used. Free dye can be accumulated and redistributed in acid-fast mushroom spores showing that this phenomenon is not peculiar to mycobacteria.—*The Nature of Acid-Fastness, D. Yegian & R. J. Vanderlinde, J. Bact., December, 1947, 54: 777.— F. G. Petrik*)

**Proteins of the Tubercle Bacillus.**—The purpose of this paper is to provide data from electrophoresis experiments concerning the insoluble residue of tuberculin, this being the fraction left after extraction by water and alcohol-ether mixtures. The extraction and electrophoresis methods are described in detail. The principle fractions found in the residue mixture were identified as nucleic acid, a protein, and a carbohydrate. The protein has a very high tuberculin activity.—*Physicochemical Analysis of Protein Antigens Isolated from the Tubercle Bacillus, II, A. Grönwall, Upsal läkaref. förh., June 30, 1947, p. 227.—(R. W. Clarke)*

**Passive Transfer of Tuberculin Sensitivity.**—Homologous passive transfer of tuberculin sensitivity was accomplished with the peritoneal and splenic cells of guinea pigs infected with BCG strain of *Mycobacterium tuberculosis*. Intense reactions were elicited in the recipients with dilution of Old Tuberculin as high as 1:100. Animals receiving splenic cells were usually less sensitive. The duration of the tuberculin sensitivity was for only a few days and was accomplished only with living cells and after an incubation period of two to three days. This suggests that the sensitivity may be principally due to antibody elaborated by such cells during their residence in the recipient.—*The Tuberculin Reaction: I. Passive Transfer of Tuberculin Sensitivity with Cells of Tuberculous Guinea Pigs, W. F. Kirchheimer & R. S. Weiser, Proc. Soc. Exper. Biol. & Med., October, 1947, 66: 166.—F. B. Seibert*)

**Passive Transfer of Tuberculin Type Hypersensitivity.**—The tuberculin type of hypersensitivity was transferred passively to normal, unsensitized guinea pigs by injection of the cells of peritoneal exudate, lymph nodes, spleen and whole blood of tuberculin-sensitive guinea pigs. The passively conferred sensitivity lasted four or five days. Positive results, employing suspensions containing 95 per cent of lymphocytes, suggest that this cell type may be of great importance in the transfer of sensitivity.—*Passive Cellular Transfer of the Tuberculin Type of Hypersensitivity, A. B. Stavitsky, Proc. Soc. Exper. Biol. & Med., February, 1948, 67: 225.—F. B. Scibert*)

**Lympholysis by Tuberculin.**—Mature lymphocytes from the blood or spleen of normal mice are not lysed by tuberculin or by the soluble substances derived from cultures of *S. enteriditis*. Lymphocytes from mice infected with tuberculosis or *S. enteriditis* are lysed specifically by the respective products from these organisms. Lympholysis of mouse cells can be blocked by adequate amounts of phosphate buffer at pH 7.2, by 0.5 per cent bovine albumin in saline, or by saline alone. Lympholysis of tuberculous guinea pig cells can be blocked by phosphate buffer or in complement-poor suspending fluids. Mouse granulocytes, monocytes and acute splenic tumor cells are not affected by the specific lytic agent before or after lympholysis. In the guinea pig, lympholysis is followed by destruction of granulocytes and monocytes as well as some of the remaining lymphocytes. Specific lympholysis can also be demonstrated in fresh heparinized mouse or guinea pig blood.—*Lytic Effect of Bacterial Products on Lymphocytes of Tuberculous Animals, C. B. Favour, Proc. Soc. Exper. Biol. & Med., June, 1947, 65: 269.—F. B. Seibert*)

**Effect of Pneumotropic Virus on Pulmonary Tuberculosis in Mice.**—Healthy mice may harbor in their lungs one or more viruses in a

latent state. A second infection may serve to provoke or activate these agents and thus be affected by their presence. Experimental tuberculosis in mice might thus be influenced by the concurrent development of infection with a latent virus. To determine the effect of active virus on the course of experimental tuberculosis (pulmonary), mice were infected with both virus and bacilli. The infecting bacillus was H37Rv given intraperitoneally. The viruses used were a strain of pneumonia virus of mice (PVM15) and an influenza A virus (PR8), both administered intranasally. Two strains of albino mice, both similarly susceptible to tuberculous infection, were employed. In the first series of experiments the virus and the bacilli were given almost simultaneously, the virus in various strengths and the bacilli usually in the same amount. A control group received no virus. At the end of 3 weeks all surviving mice were killed. It was found that of the animals receiving tubercle bacilli alone, 17 per cent showed pulmonary lesions (score ratio = 0.061), whereas of those receiving tubercle bacilli and a full dose of PVM, 73 per cent showed definite tuberculous lung lesions with a score ratio of 0.329. A tenfold decrease in the amount of the virus inoculated did not decrease the effects of the viral infection on the course of the tuberculous infection. A decrease of 100-fold in the amount of virus inoculated caused some decrease in its effect which was, however, still substantial. The influenza virus was also found to enhance pulmonary tuberculosis in mice in a manner analogous to infection induced with PVM. The viruses employed were diluted in broth containing inactivated horse serum. The intranasal instillation of the diluent alone apparently had no enhancing effect on the pulmonary tuberculosis, but when fresh human serum was used there was much enhancement of the pulmonary lesion. It seems probable that this activates the PVM latent in the mouse lung. In another set of experiments the virus was not administered to the mice until three weeks after infection with tubercle bacilli. As it was necessary that the animals live longer than in the previous experiment the dosage of tubercle bacilli was considerably less. The overall length of the experiment was six weeks. Even under these conditions the instillation of PVM or PR8 increased both the incidence and extent of pulmonary tuberculous lesions. When the inoculum of tubercle bacilli was very small neither virus caused any definite alteration in the course of the tuberculous process. Microscopic examination showed no significant qualitative differences between the tuberculous lesions in the lungs of mice which received tubercle bacilli alone or tubercle bacilli plus the viruses. However, striking quantitative differences were observed as indicated by the scoring. There was no doubt of the tuberculous nature of the lesions seen for, besides their typical histological appearance, tubercle bacilli were often present in tremendous numbers. The lesions induced by either of the viruses are very different from those caused by tubercle bacilli and can be readily recognized. The course of pulmonary tuberculosis in the mouse appears to be accelerated as a result of concurrent infection of the lung with either of the two pneumotropic viruses studied. This effect is obtained with virus inocula sufficiently small as to induce little or no definite viral pneumonia.—*The Enhancing effect of Concurrent Infection with Pneumotropic Viruses in Pulmonary Tuberculosis in Mice, M. Volkert, C. Pierce, F. L. Horsfall, Jr. & R. J. Dubos, J. Exper. Med., September, 1947, 86: 203.*—(*J. S. Woolley*)

**Recovery in Experimental Friedländer's Bacillus Pneumonia.**—Recent studies of the mechanism of recovery in pneumococcal pneumonia have shown that "surface phagocytosis" can bring about destruction of pneumococci in the lung in the absence of type-specific immune bodies. The present investigation deals with Friedländer's bacillus pneumonia in rats. Friedländer's bacillus and the pneumococcus are both encapsulated organisms, the former is Gram-negative and the latter Gram-positive, and their immune reactions are similar. This, the first of three papers dealing with the mechanism of recovery in experimental Friedländer's pneumonia, discusses the evolution of the disease. Pneumonia was produced in white rats by the

intrabronchial inoculation of the bacilli suspended in mucin. The pneumonia was lobar in type, was almost universally fatal, and simulated the acute form of the natural disease in human beings. The pathogenesis of the pneumonic lesion was studied by examination of the lungs of animals killed at frequent intervals during the course of the infection. Histologically the various stages of the pneumonia were essentially the same as in experimental pneumococcal pneumonia except for the following differences: (*1*) In isolated areas in Friedländer's pneumonia many more bacteria were encountered in the alveoli than were ever noted in the pneumococcal pneumonia. (*2*) Abscess formation was common in the late stages of Friedländer's infection, whereas it was not noted in the pneumococcal lesion. (*3*) Organization of the alveolar exudate, rarely observed in experimental pneumococcal pneumonia, was a prominent feature of the pneumonia due to the Friedländer's bacillus. The mechanism of spread of the Friedländer's lesion appeared to be the same as that of pneumococcal pneumonia, *vis.*, by way of infected edema fluid. Likewise there was noted the same phagocytosis of organisms in the lungs of even bacteremic animals dying of the infection.—*Studies on the Mechanism of Recovery in Pneumonia Due to Friedländer's Bacillus: I. The Pathogenesis of Experimental Friedländer's Bacillus Pneumonia, L. Sale, Jr. & W. B. Wood, Jr., J. Exper. Med., September, 1947, 86: 239.*—(*J. S. Woolley*)

**Recovery in Experimental Friedländer's Bacillus Pneumonia.**—Both clinical and experimental studies indicate that infections due to Friedländer's bacillus may be favorably affected by sulfonamide chemotherapy. The present report (part II of the studies on recovery) deals with the effect of such chemotherapy upon the pulmonary lesion in experimental Friedländer's pneumonia in rats. Sulfadiazine and sulfamerazine were administered through a blunt cannula by way of the mouth. When treatment with either of these drugs was begun 6 hours after inoculation, more than 90 per cent of the animals survived this otherwise fatal form of pneumonia. When treatment was delayed as late as 18 hours none of the animals survived. In those recovering most of the pneumonic lesions cleared completely, but an occasional animal exhibited small residual abscesses in the previously consolidated lungs. The recovery process in the lungs was studied histologically at various intervals during the therapy. As in the case of pneumococcal pneumonia, the principal action of the sulfonamide was upon the bacteria in the zone of advancing edema at the periphery of the pneumonic lesion. The drug appeared to stop the spread of the pneumonia, and the Friedländer bacilli were ultimately ingested and destroyed by the phagocytic cells in the alveolar exudate. Once phagocytes have accumulated in sufficient number in the infected alveoli, they destroy the bacilli by the same phagocytic process that operates in the zone of consolidation in untreated animals. The phagocytosis of bacteria in the lungs is apparently unrelated to the presence of antibody (opsonin) in the blood, for it occurs before antibody has had time to develop.—*Studies on the Mechanism of Recovery in Pneumonia Due to Friedländer's Bacillus: II. The Effect of Sulfonamide Chemotherapy upon the Pulmonary Lesion of Experimental Friedländer's Bacillus Pneumonia, L. Sale, Jr., M. R. Smith & W. Barry Wood, Jr., J. Exper. Med., September, 1947, 86: 249.*—(*J. S. Woolley*)

**Recovery in Experimental Friedländer's Bacillus Pneumonia.**—As reported in part II, encapsulated Friedländer's bacilli are destroyed by leucocytes in the pneumonic lungs of rats in the absence of circulating or local antibody. In a series of delicate experiments it is now shown that this is brought about by the same "surface phagocytosis" which operates in the case of the Type I pneumococcus under similar conditions. Direct observation of the phagocytic process reveals that leucocytes in the lung can phagocytose unopsonized Friedländer's bacilli only by trapping them against the surfaces of alveolar walls, or by pinning them against the surfaces of ad-

jacent leucocytes. Evidence is presented that the bacilli thus phagocytosed are rapidly killed in the cytoplasm of the phagocytic cells. The failure of prolonged chemotherapy to cure the lung abscesses that not infrequently complicate Friedländer's pneumonia is apparently due to the facts that, in the absence of normal alveolar walls, surface phagocytosis is relatively inefficient, and in addition the absence of oxygen renders the phagocytes sluggish or even nonviable.—*Studies on the Mechanism of Recovery in Pneumonia Due to Friedländer's Bacillus: III. The Role of "Surface Phagocytosis" in the Destruction of the Microorganisms in the Lung, M. R. Smith & W. B. Wood, Jr., J. Exper. Med., September, 1947, 86: 257.*—(*J. S. Woolley*)

**Bacteriophage for *Mycobacterium smegmatis*.**—A bacteriophage specific for *M. smegmatis* was isolated from samples of compost and soil by specific enrichment with a heavy washed suspension of *M. smegmatis*.—*A Bacteriophage for Mycobacterium smegmatis, Grace M. Gardner & R. S. Weiser, Proc. Soc. Exper. Biol. & Med., October, 1947, 66: 205.*—(*F. B. Seibert*)

**Insulin Tolerance in Amyloidosis.**—Insulin tolerance tests were given to 7 patients with amyloidosis and 7 patients with cirrhosis, all tuberculous, in an attempt to determine if hepatic dysfunction were the sole cause of insulin resistance and impaired response to hypoglycemia usually demonstrable in amyloid disease. Although the effects of impaired nutrition and/or infection could not be definitely ruled out in influencing the results, insofar as similar insulin resistant curves were demonstrated in both groups, the conclusion is warranted that in amyloid disease impairment of liver function is responsible for the increased insulin resistance.—*The Insulin Tolerance Test in Amyloidosis, S. London, Quart. Bull. Sea View Hosp., April, 1947, 9: 137.*—(*P. Q. Edwards*)

**X-Ray Cataloguing.**—This is a description of the system employed in the Department of Roentgenology at the University of Michigan for cataloguing permanent X-ray records in order to facilitate their profitable use for research and teaching. Indexing and cataloguing is carried out at the time of each patient-visit and, since the observations are to be translated into a numerical code, the tabulation provides a clear picture of their actual practical importance and forces the radiologist to avoid loose, meaningless, ambiguous statements and to phrase his comments in terse, meaningful English. The roentgenological findings are broken down into ten anatomical divisions. All statements are filed in ten diagnostical categories, and within each category all entries are segregated in separate varieties by means of punch cards and sorting machines. At the end of each year, the catalogued material is printed by a mechanical tabulating machine, 50 entries to a page, and bound into four volumes. For each individual patient, a separate coded entry is made; an asterisk is used to indicate a case of "special interest". For cataloguing these cases, Special Interest pages are provided in the index; carbon copies of the original X-ray findings follow the index in loose-leaf form.—*Cataloguing X-Ray Experiences, F. J. Hodges, Publ. Health Rep., August 1, 1947, 62: 1129.*—(*O. Pinner*)

**Abnormal X-ray Findings.**—The authors warn against confused thinking in mass radiographic programs. Pulmonary tuberculosis should not be diagnosed on the basis of one original X-ray film alone. If no tubercle bacilli are found the diagnosis should be "suspected tuberculosis", and if, in addition, the tuberculin test is negative, other reasons than tuberculosis must be found even for strongly suggestive shadows, including cavities. A provisional guide for disposition of persons with abnormal pulmonary X-ray findings, based on recent extensive experience in the United States and Denmark, is presented for use by the Tuberculosis Control Division, U. S. Public Health Service. Prerequisites for proper diagnosis are: (*1*) examination for tubercle bacilli; (*2*) identification of cavity on a 14″ x 17″ film, classifying lesions as present,

suspected, or absent; (3) tuberculin test; (4) evaluation of principal symptoms, i. e. temperature elevation, fatigue, and loss of weight. The routine procedure to be used during the one to three visits necessary to obtain and evaluate the four types of findings is described. The Guide shows nine principal groups into which the results of a person's examination may fall according to the presence, absence, or coexistence of the several findings, and outlines, for each of these groups, the follow-up and disposition of cases. The same methods are used for contacts with abnormal X-ray findings, whereas different procedures are used for follow-up and disposition of those contacts who have no abnormal X-ray findings. No person is told that he has tuberculosis unless a positive tuberculin test and bacillary findings have corroborated the "suspected tuberculosis" on the film. No person merely suspected is sent to a sanatorium. Even if he has symptoms, he can be observed in a clinic or local hospital. Thus he is spared psychological trauma and the risk of unwarranted hospitalization, and scarce sanatorium beds are reserved for infectious cases.—*Guide for Disposition of Persons With Abnormal Pulmonary Findings on X-ray Films, H. E. Hilleboe & J. Holm, Journ.-Lanc., June 1947, 67: 234.—(O. Pinner)*

**Radiological Differentiation between Pericardial Effusion and Cardiac Dilatation.**—In spite of the growing knowledge of the morbid anatomy and pathological physiology of the pericardium, the diagnosis of pericardial effusion has remained the perplexing problem that it has always been. In the author's experience two stages of pericardial effusion are not readily diagnosed: the early fluid accumulation which does not as yet display the classical symptoms, and the extreme filling of the pericardial sac, which must be differentiated from the heart enlargement due to rheumatic involvement, thyrotoxicosis, myxedema, and various types of mycardial damage. In reference to the early roentgenologic recognition of pericardial effusion, certain statements are generally accepted: (*1*) that smaller amounts of fluid, up to 250 or 300 cc., are usually not detectable fluoroscopically or on films; (*2*) that with the means available today it is not possible to differentiate between the heart shadow and the pericardial fluid shadow on the basis of difference in density. Even these fundamental rules will have to be revised, since with modern electric devices the limit of our perceptibility is constantly advanced and percussion and auscultation become the tools of another age. The author investigates, to good advantage, the value of the Valsalva test, apparently not previously applied to the problem. The dilated heart shows more marked changes in size and shape than the heart enclosed in pericardial fluid or even the normal heart. In changing from the Valsalva to the Mueller test, the weak pulsations of the dilated heart increase and become visible. In the presence of pericardial effusion, such pulsatory changes do not occur in equal measure. Observation of the angle of the bronchial bifurcation is further suggested as a diagnostic aid. Its visibility is frequently obscured in pericardial effusion. Visibility and widening to 100° to 130° favor a diagnosis of auricular enlargement or hilus tumor. Absence of lateral and posterior displacement of the esophagus, or a rather shallow, wide curved pressure zone along the whole length of the lower esophagus favors the diagnosis of pericardial effusion. Lateral and posterior displacement and localized auricular impression favor cardiac enlargement. Diminution or absence of cardiac pulsation is an important diagnostic sign of pericardial effusion, demonstrable by kymography.—*Radiological Differentiation between Pericardial Effusion and Cardiac Dilatation, J. Arendt, Radiology, January, 1948, 50: 44.—(G. F. Mitchell)*

**Hilar Densities Simulating Neoplasms.**—The value and importance of serial roentgenograms in establishing a diagnosis of pulmonary carcinoma after making use of all informative aids, such as history and bronchoscopy, is stressed. Hilar densities as found in single posteroanterior chest films is the chief concern of the author. Films showing hilar den-

sities of central pneumonia, Loeffler's eosinophilic pneumonia, primary hilar tuberculosis, lung abscess, and delayed resolution of pneumonia, all simulating bronchogenic carcinoma, are discussed. Roentgenograms in such cases may pattern themselves very closely after the early stages of the hilar type of bronchogenic carcinoma including, as well, progression into cavernous and pneumonic types. The radiologist, when interpreting hilar densities of this type, should not make an etiologic diagnosis on the basis of a single roentgenogram, since there are no criteria pathognomonic of the neoplastic lesion described in this paper.—*Hilar Densities Simulating Neoplasms, C. Gottlieb & H. S. Sharlin, Radiology, January, 1948, 50: 57.*—(*G. F. Mitchell*)

**Friedländer's Pneumonia in Infancy.**—Pneumonia caused by *K. pneumoniae* occurs with extreme rarity in infants and children. Only 4 cases have been reported in the literature. There are two possible types of lung infection: a lobar form very much like pneumococcus pneumonia, and a chronic form similar to tuberculosis. The chronic form may follow the acute infection. Diagnosis is usually a bacteriologic matter, and is usually a surprise. A case is reported of an undernourished infant who had required resuscitation at birth. On the third postnatal day it was noted to be doing badly, and to have a thick yellow mucoid discharge from the nose and throat. On the nineteenth day, inspiratory râles were heard at the apices, a leucocytosis was present, and an X-ray film showed clouding in the apices and globular densities in the right mid-lung and left basal areas. Penicillin and sulfadiazine were given for several days, and the râles cleared. In a few days a fever began, and the medication was repeated without effect. At the same time a culture from the nose and throat revealed *K. pneumoniae*. Streptomycin was immediately started in place of the other drugs, 0.67 g. daily in 8 divided doses, and this was continued for ten days. The fever dropped at once, the clinical condition began to improve steadily, and the X-ray densities began to clear. The cultures became negative in two and a half weeks, the râles cleared somewhat, but large cystic rarefactions became visible in both lung fields by radiography. These "cavities" were still present a month after the cultures became negative, and while the infant was home in apparently good health. The more frequent use of cultures in the pneumonias of infancy will doubtless reveal more cases, and allow a selective use of the available drugs.—*Friedländer's Pneumonia in Infancy, B. W. Miller, H. W. Orris & H. H. Janis, J. Pediat., November, 1947, 31: 521.*—(*W. H. Oatway, Jr.*)

**Staphylococcal Pneumonia.**—Primary staphylococcal pneumonia is one of the gravest forms of staphylococcus infection in childhood. The authors report 55 sporadic fatal cases with necropsies in children under the age of 7 years, seen in a general hospital; two-thirds were under 6 months of age. A series of 16 fatal cases in a nursery epidemic also are reported. The epidemic form often is associated with outbreaks of virus influenza. The sporadic form is predominantly a children's disease, occurring particularly during the neonatal period. In the 55 sporadic cases, pulmonary consolidation was the predominant feature in 45; in the other 10 cases, the pneumonia was associated with suppurative bronchitis and bronchiolitis; there was acute bronchiectasis in 2 and pulmonary abscess in 3 cases. The consolidation varied from a dark-red hemorrhagic pneumonia simulating the lobar type both in appearance and distribution to a diffuse gray pneumonia with suppurative softening which sometimes amounted to a localized abscess. The simple type was seen in 10 cases; in these, the duration of illness had been less than two days. If the illness had lasted longer, it was common to find early suppurative changes. Fibrinous pleurisy was seen in 16 and empyema in 26 cases. The empyema was usually unilateral. Pyopneumothorax was seen in only one and purulent pericarditis in 2 cases. There were no metastatic abscesses. The microscopic changes varied in accordance with the macroscopic. Where the condition grossly was

mainly hemorrhagic consolidation, microscopic examination showed groups of alveoli packed with red cells. At the margins, alveoli contained fibrin-free edema fluid with occasional phagocytes and clusters of gram-positive cocci. In cases which had survived a few days, there were suppurative foci, often with bronchitis, bronchiolitis and extensive infiltration and destruction of the bronchiolar walls. The nursery outbreak involved mostly premature infants. The children were healthy at birth but many mothers and nurses had influenza. They underwent sudden collapse a few days after birth and died in twenty-four to forty-eight hours. The pneumonia was diffuse in 12 and localized in 4 cases. Of the 12, 6 were hemorrhagic, 5 had ordinary non-suppurative bronchopneumonia and 1 had multiple abscesses. The other 4 also had multiple abscesses. None had empyema. The bronchi were free of inflammation and exudation. Microscopically, there were areas of hemorrhagic and suppurative pneumonia with purulent bronchitis and bronchiolitis. Staphylococci were found in the throats of many nurses. The milk supply was found to be uncontaminated. The infection is bronchogenic in origin. Diagnosis is difficult. Valuable time is lost in waiting for the uncertain results of bacteriological studies. The authors recommend immediate institution of oral and intramuscular penicillin therapy, supplemented by sulfadiazine and oxygen.—*Staphylococcal Pneumonia in Childhood, K. J. Guthrie & G. L. Montgomery, Lancet, November 22, 1947, 2: 752.*—(*A. G. Cohen*)

**Meningococcal Pneumonia.**—This is a rare condition. In one month, 2 cases were seen. There was no meningitis in either case. In one, meningococci were found in the bloodstream, but not in the sputum. In the other, the organism was recovered from both sputum and pleural fluid.—*Meningococcal Pneumonia, I. B. Brick, New England J. Med., February 26, 1948, 238: 289.*—(*A. G. Cohen*)

**Therapy of Pertussis and Pneumonia.**—Twenty-six infants with uncomplicated pertussis were given hyperimmune gamma globulin (2.5 cc. daily for four days intramuscularly); no case developed pneumonia, no case died, and two-thirds had a good or excellent response. Twenty-six infants with pertussis and pneumonia were treated with the globulin and with 130 mg. of sulfadiazine per kilogram per day by mouth; most of these were very ill when first seen. Two died (7.7 per cent), but neither death could be attributed to failure of therapy. The recovery of the others was often dramatic.—*The Treatment of Pertussis and Pneumonia Complicating Pertussis: The Role of Hyperimmune Gamma Globulin and Sulfadiazine, H. Brainerd, J. Pediat., January, 1948, 32: 30.*—(*W. H. Oatway, Jr.*)

**Streptomycin for H. Influenzae Empyema.**—Pneumococci cause 75 to 80 per cent of empyemas, and staphylococci and streptococci account for all but 1 per cent of the remainder. *H. influenzae* is one of the rare pleural invaders, and only a few instances of streptomycin therapy have been reported. The case is reported of a girl, aged ten years, who was seen a month after the onset of a respiratory illness with pneumonitis, empyema, mild cardiac decompensation, and evidences of cerebral and renal embolism. After a good initial response to penicillin the influenza bacilli became resistant. Large doses of streptomycin (250,000 units every three hours for a total of 20 gm.) were given, and 500,000 units were left in the pleural space after each of eight thoracenteses. The empyema decreased in amount and became sterile, but the fever and clinical condition did not improve until about three weeks after the drug was discontinued. The child then became well during the ensuing three months.—*Streptomycin Treatment of Empyema Caused by Hemophilus Influenzae, A. J. Fisher & E. B. Shaw, Am. J. Dis. Child., October, 1947, 74: 468.*—(*W. H. Oatway, Jr.*)

**Viral Pneumonia.**—*Primary atypical pneumonia* is the term used to signify the type of viral pneumonia which has been prevalent in recent years. The cause has not been definitely established, but it is believed to be a

filter-passing agent, presumably a virus. Pathologic data are relatively meager because the case mortality rate has been so low. An acute bronchiolitis and interstitial pneumonitis have been noted. Pleural surfaces were smooth. The amount of edema or hemorrhage in the parenchyma showed great variation. The microscopic picture was variable. Primary atypical pneumonia appears to be transmitted from man to man by the respiratory route. The incubation period is usually ten to fourteen days with a range of five to twenty-one days. The disease appears to be moderately communicable. Epidemics tend to run a period of many months. All age groups are affected. Sporadic cases occur at frequent intervals in the general population. The duration of immunity following one attack has not been definitely established. The onset is usually gradual. The most common symptoms are fever, chills, cough, malaise and headache. Coryza and sore throat are less common. Chest pain is often noted. Physical examination is usually unremarkable except that the patient appears somewhat ill, the pharynx is often moderately injected, and chest examination often reveals small areas of dulness and subcrepitant râles. The subsequent course is quite variable. Most patients never appear severely ill. For each patient who develops pneumonia, there are three to ten times as many who have the virus infection, but who do not develop pneumonia. Only about one-fifth of the patients with pneumonia become seriously ill, with prolonged fever and extensive pulmonary infiltration. The white blood count is usually within normal limits. Counts exceeding 12,000 are exceptional unless there is a concurrent bacterial infection. As the disease progresses, however, the white count tends to rise. The sedimentation rate is elevated early in the course of the pneumonia and returns gradually to normal. Chest X-ray films reveal areas of increased density compatible with pneumonia. Fifty-six per cent of 801 patients with a clinical diagnosis of primary atypical pneumonia reveal significant titers of cold agglutinins in the convalescent sera. Comparable titers (40 or over) were found in only 4.4 per cent of 1,719 patients with other diseases, and in 8.1 per cent of 209 normal subjects. The agglutinin titer, in general, begins to rise between the seventh and fourteenth days after onset, reaches a peak about three weeks after onset, and then falls off fairly rapidly. In certain patients titers remain significantly elevated for only a short period. Blood serum had best be examined during the first week of the illness and one or more specimens collected after the twelfth day of the disease. The test has considerable practical value as a means of classifying cases of primary atypical pneumonia. Using the streptococcus MG agglutinins, Horsfall found that 48.9 per cent of 438 cases of primary atypical pneumonia showed titers of 20 or more, while only 5.1 per cent of 349 patients with other diseases and 5.3 per cent of 357 normal subjects showed titers as high as this. In general, patients developing streptococcal agglutinins also develop cold agglutinins, but many patients with the latter do not have the former in significant titer. For this reason the cold agglutination test appears to be more useful for diagnosis. The significance of cold agglutinins and other streptococcal antibodies is not yet clear. *Treatment:* Therapy remains symptomatic. Sulfonamides and penicillin have no definite therapeutic effect. Treatment with convalescent serum has not been proved effective. Codeine is useful for patients with distressing cough. Oxygen may be necessary for severely ill patients. Convalescence may often be long. *Viruses in the psittacosis group:* This virus is present not only in psittacine birds, but also in pigeons, doves, domestic poultry, sea birds, and probably other avian species. The latent virus has been recovered from rodents and cats. The number of human cases of pneumonia caused by psittacine viruses is relatively small. Pneumonia caused by the psittacosis virus is often much more severe than ordinary primary atypical pneumonia. Psittacosis pneumonia may have a case fatality rate of between 20 and 40 per cent. It is important to note that experimentally the psittacosis group of viruses are susceptible to penicillin

and preliminary clinical trials in humans have been definitely encouraging. Clinically the patient is sicker than with a simple pneumonia. The white count is usually low, although considerable variation occurs. X-ray findings are indistinguishable from those seen in other viral pneumonias. Diagnosis can be established by isolation of the agent from sputum or, in early cases, the blood, or by demonstrating a greater than four-fold increase in antibody titer in compliment fixation tests with acute and convalescent sera. There is no significant increase in either cold or streptococcus MG agglutinin titer. Treatment should be instituted with penicillin as soon as the diagnosis appears resonably well founded on clinical grounds since laboratory confirmation cannot be expected at an early date. At least 50,000 units intramuscularly every three hours should be used. *Influenzal pneumonia:* Either type A or type B influenza virus may cause a primary viral pneumonia, or may initiate bacterial or mixed viral and bacterial pneumonia. There is at present no specific treatment for primary influenza viral pneumonia. Secondary bacterial pneumonia responds favorably to sulfonamide or penicillin therapy. *Q fever:* This rickettsia may be responsible for a type of pneumonia virtually indistinguishable from the ordinary viral pneumonia. Clinically the cases are indistinguishable. No specific therapy is available. Diagnosis rests on laboratory means.—*Viral Pneumonia, G. Meiklejohn, M. Clin. North America, November, 1947, 31: 1442.*—(*B. Hyde*)

**Pneumonia of Azygos Lobe.**—A case report of an 18-year-old male with pneumonia of the azygos lobe, which resolved on a full course of sulfadiazine, is presented. Pulmonary tuberculosis, apical segmental consolidation, segmental atelectasis, mediastinal effusion, and mediastinal neoplasm were considered in the differential diagnosis.—*Pneumonia of the Lobe of the Azygos Vein, T. C. Studdert & A. K. Turnbull, Brit. J. Radiol., March, 1947, 20: 119.*—(*B. Hyde*)

**Treatment of Pneumonia.**—*Lobar pneumonia*—abrupt onset with chill, fever, cough, sharp chest pain, dyspnea, rusty or blood-streaked sputum. There may be splinting of the affected side of the chest, pleural friction rub, dulness to percussion and tubular breathing. Chest X-ray reveals a dense, homogeneous shadow. Pneumococci are the causative organisms in 97 per cent of attacks of lobar pneumonia. Sulfadiazine is the sulfonamide of choice in therapy. Dosage may be 4 g. followed by 2 g. in two and four hours, followed by 1.0 g. every four hours day and night until temperature reaches normal and remains there for forty-eight to seventy-two hours. Reactions may occur in 10 per cent of patients. To prevent renal complications, patient should receive 3,000 centimeters of fluids daily; also 2 g. of sodium bicarbonate three times a day. Other toxic reactions are skin reactions, fever and blood changes. Penicillin is effective against pneumococci as well as other gram positive organisms. Dosage may be 20,000 units every three hours of the aqueous solution, or a daily injection of penicillin beeswax and oil, 200,000 to 300,000 units. Penicillin may be given orally, an initial dose of 200,000 units, followed by 100,000 units every three hours. Results of chemotherapy are clear. Before specific therapy, the mortality rate at Cook County Hospital, Chicago, was about 35 per cent (24 to 48 per cent). With chemotherapy, the mortality rate has dropped to 12 per cent (40 per cent of these were patients who were moribund on admission). Antipneumococcus serum may be used if sputum pneumococci are typed. It may be used with chemotherapy in cases where the prognosis is dubious. *Atypical pneumonia* occurs more frequently in adolescents and young adults. The lungs may be clear for several days. The leucocyte count tends to be normal. Chest X-ray may reveal only increased hilar markings with possible irregular shadows, or there may be more extensive mottling. A virus origin is likely. There is no specific therapy. Bed-rest and symptomatic care are the rule. Sulfadiazine and penicillin often are used and results may be most gratifying. *Bronchopneumonia* is usually secondary to other infections. It is usually bilateral. Penicillin is usually more effective than sulfadiazine. Ex-

pert nursing care is necessary. If the causative organism is the beta hemolytic streptococcus, sulfadiazine should be used, and continued for at least one week of normal temperature. *Aspiration pneumonia*—there is a necrotic as well as an infectious process present. The cough reflex is diminished or absent. This may occur in coma and the symptoms may be profound. Prophylaxis is important since the prognosis is grave. Penicillin is used for therapy. *Friedländer pneumonia* may be lobar or lobular. Sulfadiazine and streptomycin are most effective in treatment.—*The Treatment of Pneumonia, F. B. Kelly, M. Clin. North America, January, 1947, 31: 52.*—(*L. Hyde*)

**Penicillin for Juvenile Respiratory Infections.**—One hundred and forty-three infants and children with acute respiratory infections, and with fevers of at least 100° F. on admission to the hospital, were given penicillin by mouth. The drug was crystalline type G, unbuffered. The dosage was varied according to age, but averaged 3,000 U. per kilogram body weight, and was given every three hours. The blood levels were satisfactory and well maintained in children from one to 12 years of age; they were higher in infants of less than one year. Two-thirds showed good results, *i.e.*, subsidence of fever and clinical improvement within forty-eight hours. A virus component was probably present in many cases. Evaluation on the basis of cultures or blood counts changed the results only slightly. The method is considered to be valuable.—*The Treatment of Acute Respiratory Infections in Children with Orally Administered Unbuffered Penicillin Solutions, W. S. Hoffman, J. W. Hofer & H. Gordon, J. Pediat., January, 1948, 32: 1.*—(*W. H. Oatway, Jr.*)

**Inhalation of Penicillin Dust.**—The principle utilized in this newer method is based on the negative pressure created during normal inspiration. It is felt that inhaling penicillin dust in this manner is more physiological and permits a more even and perhaps wider distribution of medicament throughout the respiratory tract than by the use of solution nebulized by oxygen pressure. The patient is not required to manipulate an exhaling valve during the expiratory phase, the cumbersome equipment of oxygen tank and gauge is not necessary and the preparation of solutions is obviated. The inhalation of the dust particles yields a greater effect than the vapor because of the increased concentration of penicillin per unit area and because of the localized absorption of the penicillin where it comes in contact with the mucous membranes. The authors used crystalline sodium penicillin processed to # 50–100 mesh particles and present details of an oronasal mask similar to the simple respirator used by spray painters. All patients received 100,000 units of penicillin dust by inhalation for twenty minutes three times a day. No sensitivity reactions were noted in 68 patients whose diagnoses varied from common colds to bronchiectasis. Blood level curves indicated a slow absorption, the maximum level being obtained about three hours after inhalation. Clinical improvement was noted much more quickly than had been observed previously in a similar group of patients with penicillin aerosol, and fewer treatments were necessary for maximum benefit.—*Inhalation of Penicillin Dust, L. Krasno, M. Karp & P. S. Rhodes, Science, September 12, 1947, 106: 249.*—(*E. A. Rouff*)

**Intrabronchial Penicillin in Suppurative Disease.**—The authors decided to treat cases of bronchopulmonary suppuration by direct instillation of penicillin. They used the same catheters as for bronchography. A total of 63 patients were treated. Accurate localization of the lesions is necessary. Following local anesthesia, the catheter was directed toward the bronchus of the diseased segment. The patient was then placed in such a position that the end of the catheter was vertically above the affected segment. A solution of 100,000 units of penicillin in 5 to 10 cc. of saline was injected slowly. The patient remained in position for thirty minutes. The treatment was repeated every two days. The average number of treatments was 8. There was no selection of cases. In those with underlying disease such as bronchiectasis or neoplasm, there was no definitive cure but considerable

relief of symptoms was obtained. In cases of lung abscess, there was complete recovery in 17, good clinical results without cure in 6 and failures in 5 cases. Four patients with suppurative bronchitis were improved. The amount of sputum decreases after the first installation and almost disappears after the second. Simultaneously, there is a drop in fever and the patient begins to gain weight.—*Treatment of Broncho-Pulmonary Suppuration by Local Injection of Penicillin, H. Metrás & J. Lieutier, Thorax, December, 1947, 2: 196.*—(*A. G. Cohen*)

**Aerosol Penicillin in Respiratory Disease.**—A nebulizer must deliver particles of 5 microns in diameter or less in order to be of value in respiratory disease. A test of suitability can be made with 5 per cent glycerine which should give a fine smoke which floats in the air. In this study, 34 subjects were treated with aerosols of Zephiran (alkyl dimethyl benzyl ammonium chloride) in aqueous solution; this is a detergent. Use of detergents is advantageous in the treatment of sheltered foci since the activity of both penicillin and streptomycin is enhanced by their use. Originally an initial dose of 50,000 units of penicillin was injected intramuscularly followed by 10,000 units every three hours for ten days. Later daily injections of penicillin in beeswax were substituted. Immediately prior to treatment, 0.5 cc. of a 0.25 per cent solution of neosynephrine was administered through the nebulizer. This was followed by administration of 100,000 units of penicillin in 3 cc. of Zephiran in a 1:1000 aqueous solution. This was repeated every eight hours for ten days, during which time the patient remained in the hospital. Of 21 patients with bronchiectasis, 10 were improved, 6 were improved temporarily but relapsed and 5 were not helped. Of 12 patients with chronic bronchitis, 8 were improved, 1 relapsed after temporary improvement and 1 was not relieved. In 2 patients with pneumonoconiosis, there was definite benefit in that respiratory deficiency was relieved. Good results were obtained in one case of severe asthma and in improving the secondary infection in 2 cases of tuberculosis.—*Aerosol Therapy of Respiratory Disease, V. Bryson & E. J. Grace, New England J. Med., November 6, 1947, 237: 683.*—(*A. G. Cohen*)

**Intrabronchial Penicillin in Lung Abscess.**—Eleven cases of lung abscess were treated by intrabronchial instillation of penicillin. Treatment was given every two to three days for a total of three to eight days. On the other days, nebulized penicillin was administered. Of the 11 cases, 10 were cured.—*Intrabronchial Penicillin for Lung Abscess, E. Rosenthal, Lancet, December 6, 1947, 2: 829.*—(*A. G. Cohen*)

**Penicillin in Chest Surgery.**—A study has been made of the levels of penicillin in the blood serum after administration by various portals, and of levels in the pleural fluid after intramuscular injection. The intramuscular route is the most efficient in producing and maintaining blood levels, and it results in satisfactory levels in the pleural fluid; 40,000 units is a better dose than 20,000. Direct intra-pleural instillation produces high and prolonged local concentrations, with some absorption; it is recommended when aspirations are being done, or for postoperative inclusion. Inhalation of an aerosol obviates the use of hypodermic injections, but requires cooperation of the patient, wastes drug in producing suitable blood levels, and is inferior to intratracheal instillation for the latter effect. It should only be used, with these limitations in mind, for the temporary reduction of the Gram positive coccal flora. It is not a panacea, but does have satisfactory local efficiency; direct nebulization was superior to the use of a BLB mask.—*An Evaluation of Methods of Penicillin Therapy in Thoracic Surgery, J. H. Donnely, F. J. Phillips, J. T. Bartlett, & W. E. Adams, Ann. Surg., October, 1947, 126: 579.*—(*W. H. Oatway, Jr.*)

**Amebic Lung Abscess.**—Amebiasis is endemic in Argentina, where 20 to 30 per cent of the population is infected. Amebic lung abscess is rare and very often mistaken for tuber-

culosis, cancer or mycotic disease. The purpose of this paper is to facilitate the early diagnosis of this condition. Only occasionally is amebic lung abscess due to lymphatic or hematogenous spread. Generally, it is a direct extension from liver abscesses. First, there is hepato-pleural irritation with all the symptoms of a subphrenic abscess. After invasion of the lung, profuse sweating, tachycardia and cyanosis with the clinical findings of pneumonitis or atelectasis set in and may be mistaken for a fulminating tuberculosis of the right lower lobe. There may be temporary improvement until repeated disseminations of amebae cause severe broncho-pleuro-pulmonary irritation with persistent asthma-like dyspnea. Finally, the abscess breaks and opens into a bronchus, the mediastinum or pericardium, sometimes into the peritoneal cavity or through the chest wall. Most common is the bronchial perforation which causes sudden vomica of great quantities of odorless chocolate colored matter. This is the most important differentiation from lung abscess. The sputum may be sterile or it may contain any one of the pyogenic bacteria. Only occasionally, amebic cysts can be found. Inoculation of the sputum into the rectum of young cats may help to establish the diagnosis. In cases of proved intestinal amebiasis with negative findings for acid-fast bacilli the possibility of amebic lung abscess should be considered. However, the coexistence of tuberculosis is not uncommon. Bronchoscopy and bronchography should be done to rule out cancer. Exploratory thoracotomy is a potential means for spreading infection to the chest wall, as is diagnostic pneumothorax. All cases of amebic lung abscess show a partial or complete paralysis of the hemidiaphragm. A freely movable diaphragm is proof that there is no involvement of pleura or lungs. Lung cancer in its early stages is associated with severe pain, whereas the pain in pulmonary amebiasis is only slight. The differential diagnosis from hydatid cyst is very difficult and has sometimes to rely upon biological tests only. Tuberculosis of the right lower lobe must be ruled out by history of contact and the tuberculin reaction. In the X-ray the abscess is surrounded by a zone of inflammatory reaction. For the treatment of amebic lung abscess emetine is the specific drug. The history of a significant case is given.—*Abscesco amibiano de pulmon, M. Schneier, Arch. argent. de tisiol., 1946, 22: 42.*—(*W. Swienty*)

**Pulmonary Infarction in Children.**—Only about 100 cases have been referred to in the literature since 1833. The condition is probably unrecognized at times, since the present report from the Mayo Clinic includes 38 new cases. Embolism and thrombosis in the lung are the causes of infarction. It is difficult to distinguish between the two, but embolism probably occurs with slightly greater frequency. A local pathological change in the lung is usually required to produce the infarction. The usual factors in thrombosis are endothelial injury, circulatory stasis, and changes in the blood; the contributing causes may be infection, atelectasis, allergy, and venoclasis. In 24 of the 75 cases previously described in detail, thrombi were noted elsewhere in the body. Pulmonary and general symptoms occur following the occlusion of a pulmonary artery, the intensity depending upon the speed of obstruction. Death may be due to the mechanical changes and/or reflex factors. A diagnosis of infarction during life is rare. A sudden onset of shock with cardio-respiratory embarrassment is suggestive, but the onset may be slow, with intra-bronchial symptoms similar to those of pneumonia. In the differential diagnosis pneumonia and atelectasis must be considered. X-ray signs become visible in twelve to twenty-four hours. In the current series of 38 cases, only 3 were diagnosed clinically. The condition followed surgery in 42 per cent of the cases, a lung infection was present in 34.2 per cent, and some sort of infection was present in the body in 84.2 per cent. Children with a background similar to that which has been described should be observed closely for evidence of peripheral thrombosis; the onset of suggestive symptoms may then lead to the diagnosis.—*Infarction of the Lung in Children, E. Zuschlag, Am.*

*J. Dis. Child., October, 1947, 74: 399.—(W. H. Oatway, Jr.)*

**Hemopneumothorax in Infarction of Pulmonary Lobe.**—A case is reported of a 39-year-old colored male who developed complete infarction of the lower lobe of the left lung subsequent to long continued passive congestion. The infarction underwent rapid aseptic softening with consequent rupture into the pleural sac, producing hemopneumothorax. A search of the available literature has revealed only 5 such cases, in only 3 of which the lesion was proved by postmortem examination. To this small series the authors add a sixth case, which shows several unusual features, namely: the large size of the pulmonary infarction, the aseptic character of the softening, its rupture into the pleural cavity with the causation of hemopneumothorax, jaundice and a high blood urea nitrogen. An analysis of dates on reported cases is also given.—*Infarction of an Entire Pulmonary Lobe with Subsequent Aseptic Softening Causing Sterile Hemopneumothorax, A. J. Rawson & J. A. Cocke, Am. J. M. Sc., November, 1947, 214: 520.—(G. F. Mitchell)*

**Progressive Coccidioidomycosis.**—A 24-year-old male developed headache, fatigue and fever two weeks after he had made two trips to the San Joaquin valley. Chest X-rays, at first negative, later showed pulmonary and pleural involvement on the left. Coccidioidin skin tests were positive at the onset; later, they became negative. Complement fixation tests for coccidioides immitis remained positive at 1:8 dilution throughout. Gastric lavage specimens and pleural fluid were negative for bacteria and fungi. Penicillin therapy was ineffective in doses of 60,000 units every three hours. The course was downhill and the patient died in coma about five months after onset of symptoms. Postmortem examination revealed extensive caseous and fibrotic involvement of the lungs and pleura. There was also a generalized peritonitis. The brain revealed edema, hyperemia and focal degeneration. Microscopic examination revealed numerous coccidioides immitis organisms in the lungs, tracheobronchial lymph nodes, liver and kidneys. Most of the organisms were in the spherule stage; a few were enlarged and showed endospore formation. This patient illustrated how short a contact with infected dust can give rise to the disseminated disease. Stabilization of antibody formation as manifested in the complement fixation tests in this case is usually regarded as an indication that the infection is being adequately controlled. In the later course of the disease in this patient, the tuberculin test was positive and the coccidioidin test negative; this combination can be misinterpreted as evidence of active tuberculosis. The pathological findings indicated lymphatic and hematogenous dissemination similar to that occurring in disseminated tuberculosis.—*Progressive Coccidioidomycosis: Report of a Case, N. A. Harvey, Ann. Int. Med., March, 1948, 28: 651.—(H. R. Nayer)*

**Nocardiosis.**—A routine chest film of a 26-year-old physician revealed a minimal infiltration in the right apex. On one occasion a questionable positive sputum was reported. The lesion increased in size and pneumothorax was instituted. A transient pulmonary infiltration was treated with penicillin. The patient developed a right pleural effusion; the fluid was clear and negative for tubercle bacilli on culture and guinea pig inoculation. After several weeks purulent material was aspirated; this revealed numerous colonies of *Nocardia asteroides* in pure culture. In spite of treatment with streptomycin and penicillin the pleural fluid continued to show *N. asteroides*. Marked sensitivity of the organism to sulfadiazine could be demonstrated. The patient received 75 g. of sulfadiazine intrapleurally and 1,268 g. orally. The pleural effusion became sterile and was gradually absorbed. The patient recovered completely and has remained well during a period of observation of fourteen months.—*Nocardiosis, Nocardia Asteroides Infection Simulating Pulmonary Tuberculosis, R. P. Glover, W. E. Herrell, F. R. Heilman & K. H. Pfuetze, J. A. M. A., January 17, 1948, 136: 172.—(H. Abeles)*

**Pulmonary Aspergillosis.**—The histories of 2 patients are reported. The first patient had been treated for tuberculosis on the basis of numerous hemoptyses and pulmonary infiltrates below the left clavicle; tubercle bacilli were never demonstrated. Left upper lobectomy was carried out. The lesion was found to be a bronchiectatic cyst filled with material in which was demonstrated mycelia and spores of a fungus resembling aspergillus. Presenting symptoms of the second patient were hemoptyses and generalized macular rash. Roentgenograms revealed infiltrations in both hilar areas extending paravertebrally to both apices; later, an effusion appeared at the right base. Penicillin therapy was ineffective and this patient died one month after admission. Postmortem examination revealed considerable interstitial and parenchymatous involvement of the lung with occulsion of many pulmonary vessels and resulting tissue necrosis. Numerous fungi, identified by culture as Aspergillus fumigatus Fresenius, were found in the lungs and skin lesions. Numerous granulomatous lesions were seen in the liver and spleen. Sections of the sternum revealed almost complete aplasia of the marrow; this finding has not been previously reported as a complication of aspergillosis. The unrestricted spread of the disease in the second case contrasts with the localized process in the first patient.—*Pulmonary Aspergillosis: Report of Two Cases, B. Gerstl, W. H. Weidman & A. V. Newmann, Ann. Int. Med., March, 1948, 28: 662.*—(*H. R. Nayer*)

**Aspergillosis.**—Pulmonary aspergillosis occurs in farmers, feed mill workers, fur cleaners utilizing rye flour for removal of grease, threshers and others who have contact with birds or grains. The present case history deals with a child in whom, at the age of two years, a chest roentgenogram showed bronchopneumonia. At the age of 2.5 years and again at 4 years an inflammatory consolidation of the right lower lobe was found. A fluctuant mass developed at the lower costal margin in the left anterior axillary line. Culture of pus aspirated from that lesion revealed *A. fumigatus*. When the child was 5 years old pneumonia of the left lower lobe was found. Laboratory studies were noncontributory. At the age of 7.5 years the child developed pneumonia in both lower lobes and a cerebellar abscess from which *A. fumigatus* was cultured. The child died at the age of 8. Abscesses were found in the brain, dura, heart, lungs, mediastinum, lymph nodes, spleen, liver, right kidney and right ankle. Cultures from various sites revealed *A. fumigatus*. A hematogenous dissemination of the organism is likely.—*Aspergillosis and the Aspergilli: Report of a Unique Case of the Disease, E. P. Cawley, Arch. Int. Med., October, 1947, 80: 423.*—(*G. C. Leiner*)

**Bronchiectasis.**—Of 400 cases of bronchiectasis, 59 per cent were found unsuited for surgery because of too much or too little disease, lack of symptoms, limited cardiovascular reserve, advanced emphysema, concurrent disease or refusal by the patient. Much can be done for these patients with medical care. This is considered under the following headings: mechanical drainage, control of infection, improvement of general health and prevention. Mechanical drainage requires a suitable position. The head and thorax must extend vertically downward from a high bed or table. Secretions should be thinned out by use of steam, ammonium chloride and creosote. Depressants should be avoided. Bronchodilators should be inhaled through nebulizers. Deep expiratory and inspiratory breathing should be practiced before and during postural drainage. Drainage should be performed often enough to keep the passages empty. Bronchoscopic drainage is not indicated as a routine measure. For control of infection, aerosol and intramuscular penicillin are recommended. These are of value particularly at the beginning of an upper respiratory infection. Treatment of sinusitis also is important. Improvement of general health consists of protection against upper respiratory infections, treatment of anemia, avoidance of cold and wetness, elimination of smoking and change of climate. Prevention consists of early diagnosis and

treatment of bacterial pneumonias and re-expansion of resultant atelectatic lung tissue, especially in children; this is accomplished by deep breathing exercises.—*Bronchiectasis: Treatment and Prevention, T. L. Badger, New England J. Med., December 18, 1947, 237: 937.*—(*A. G. Cohen*)

**Pathogenesis of Bronchiectasis.**—Any theory of the pathogenesis of bronchiectasis must explain four features: (*1*) The disease is rarely diffuse. (*2*) It is usually not progressive and rarely spreads. (*3*) It is rarely an isolated finding in an otherwise normal lung; the surrounding pulmonary parenchyma is abnormal. (*4*) It is characteristically a disease of youth. The incidence of the following features in the specimen of a series of 50 lobectomies for bronchiectasis was tabulated: chronic bronchial inflammation, congenital abnormalities, bronchostenosis, atelectasis and pneumonia or pneumonitis. Bronchostenosis and congenital abnormalities were found to be unimportant except in a few cases. The other three factors were each present in more than 80 per cent of cases, but no factor was constant and none was ever a solitary finding unassociated with at least one other factor. The author concludes that bronchiectasis is an acquired disease of the pulmonary parenchyma and the bronchial tree. Parenchymal involvement varies in detail but one feature is common to all cases, namely, reduction in the number of areated alveoli, a condition which may or may not be reversible. This results in a low intrathoracic pressure which is a very effective bronchodilating force. The assumption of a primary bronchial infection with secondary atelectasis or pneumonitis fulfills the four criteria. In actual experience, bronchial obstruction causes atelectasis by obstructive secretion. This is especially true in infants and children.—*The Pathogenesis of Bronchiectasis, T. B. Mallory, New England J. Med., November 22, 1947, 237: 795.*—(*A. G. Cohen*)

**Hemangioma of the Lung.**—Visceral hemangiomata are uncommon, and their occurrence in the lung is very rare. Less than 20 pulmonary cases have been reported, half of these since the definitive description in 1938 by Rodes of cavernous hemangioma of the lung as a clinical entity. A year later the first antemortem diagnosis was made. Probably all cases are congenital, several have been multiple, and physiologically they are arteriovenous fistulas. Perhaps in some cases the shunt enlarges with age, with a resultant late development of cyanosis, though some cases are cyanotic from birth. Typical symptoms, signs, and blood changes are described. A late inspiratory, continuous murmur may be heard over the lesion; clubbing of the digits is a common sign. The X-ray films show nonprogressive, cylindrical masses, bronchograms are negative, and pulsations are seen fluoroscopically. In six previous cases which have been operated on excision of a part or all of the lung has been curative. The case of a 29 year old male is reported. Symptoms had been present for eight years, following a pneumonia, cyanosis had been noted for six years, and the condition gradually became debilitating. X-ray and laboratory findings were typical. A successful lobectomy was done, the lesion was demonstrated in an angiogram of the specimen, and the patient is well several months after surgery.—*Pulmonary Cavernous Hemangioma with Arteriovenous Fistula, Surgical Management, J. D. Bisgard, Ann. Surg., December, 1947, 126: 965.*—(*W. H. Oatway, Jr.*)

**Bronchotomy for Bronchial Tumors.**—In order to avoid resection of healthy lung tissue, bronchotomy is recommended for the removal of benign bronchial tumors in selected cases. These are cases in which there has been a relatively rapid development of obstructive symptoms, and in which there appears to be relatively little pulmonary infection. The surgical hazards are reviewed and the technique is discussed. The procedure was carried out in one case, with an excellent result.—*The Indication for Posterior Transpleural Bronchotomy in the Management of Intrabronchial Tumors, H. T. Langston &*

*R. T. Fox, Surg., Gynec. & Obst., February, 1948, 86: 192.—(A. G. Cohen)*

**Pulmonary Disease in Beryllium Workers.**—The late Dr. Leroy Gardner was intensely interested in the problem of detecting the agent responsible for the lung lesions in patients who had been exposed to beryllium compounds in the form of dust. While he was not prepared actually to condemn beryllium, he definitely felt that it was involved in some as yet unrecognized way. It was at his suggestion that the roentgen study of the chest films of 32 patients, exposed to beryllium dust and in whom delayed pulmonary changes occurred, is presented. The majority of the patients were females having an average exposure of sixteen months. An average of twenty-months transpired after the period of exposure before symptoms of the first X-ray evidence were discovered. The physical findings were limited to impaired resonance and fine râles throughout the lungs. Cyanosis was present in several cases. The laboratory findings were meagre, polycythemia being the only constant factor. Ventilation function tests revealed respiratory disability. The clinical course was variable. Some patients died after an illness of two years; many improved and were able to work though they were not symptom-free. Pathological study showed a granulomatous reaction infiltrating or completely obliterating the interstitial tissue as the predominant lesion. The mediastinal nodes were similarly involved. Four patients were thoroughly investigated and their histories are presented. The roentgen findings are limited to the lungs and are characterized by a bilateral uniformly widespread diffusely granular or "sandpaper" appearance and/or nodular infiltration. No definite disease entity is known which can produce the fine disseminated granular type of infiltration observed in these cases. In both types, but more so in the granular, coalescent or confluent shadows may develop which are more noticeable in the middle third of the lung fields. A reticular pattern may also be seen on stereoscopic examination in the granular type. The apices and costophrenic sulci were generally free. Vascular markings are obscured except at the base and in advanced cases may be obliterated. No valid correlation between the clinical course of the disease and the magnitude of the pulmonary changes could be made. The final result also is not known in those patients with clinical remission but persistent lung lesions. The prognosis should be guarded in patients with the granular type of nodulation, especially when confluent shadows are superimposed. It is also emphasized that roentgen changes may exist with insignificant or no symptomatology. Roentgen differential diagnosis may be difficult. Many of the patients were at first suspected of having Boeck's sarcoid, while a diagnosis of silicosis was almost as frequently entertained. Other pulmonary diseases that enter into consideration are chemical pneumonitis, tuberculosis, bronchomycoses, cardio-vascular disease, miliary carcinosis and erythema nodosum. It is possible that a serious type of pneumonoconiosis not heretofore described is being dealt with.—*Pulmonary Disease in Workers Exposed to Beryllium Compounds: Its Roentgen Characteristics, L. M. Pascucci, Radiology, January, 1948, 50: 23.—(G. F. Mitchell)*

**Bagassosis.**—Bagasse disease of the lungs, or bagassosis, is a pulmonary disorder brought about by the inhalation of dried bagasse dust. Only 30 or 40 cases have been reported. Most cases reported are from localized communities where the fibers of bagasse are processed into the manufacture of acoustical and thermal insulating building materials and refractory brick, or where the sugar cane is grown. The same characteristics for the disease are reported in all places. About two months' exposure to the dust are required before symptoms appear, although the time has varied from three weeks to two years. The disease manifests itself as an acute febrile illness with extreme shortness of breath, a persistent cough with scanty mucoid sputum and a profound weakness. The onset is insidious and gradual, the patients usually not realizing that they are ill until they are seized by a sudden cough-

ing spell and become so dyspneic as to be forced to rest. Patients with a long exposure to heavy concentrations of the dust become critically ill and appear to have a severe bronchiolitis and pneumonia. A mortality rate of 8.3 per cent was reported in 24 cases collected, which indicates that this is an industrial disease of a serious nature. In cases having a short exposure, the disease usually clears up in two to six months. In view of the obscurity of the exact etiology of bagassosis, there is no specific treatment. Most of the patients have been treated symptomatically. There is no notable response among those in whom the sulfonamides were tried, or in the one case treated by penicillin. Oxygen has afforded some relief of the dyspnea in the more serious cases. The pulmonary changes incited by inhalation of bagasse dust consist of diffuse infiltration and consolidation and an acute bronchiolitis or pneumonia similar to that seen in pneumoconiosis. However, in one respect it differs markedly; this is a reversible reaction with resolution and the lung regains its normal appearance on the roentgenogram. Three cases of bagassosis are reported, including a discussion of the symptoms, the physical and laboratory findings, and the clinical course.—*Bagasse Disease of the Lungs, D. V. LeMone, W. G. Scott, S. Moore & A. L. Koven, Radiology, November, 1947, 49: 556.—(G. F. Mitchell)*

**Pulmonary Hemosiderosis.**—The literature contains reports of 17 cases, all but one of which terminated fatally. All but one were children who ranged in age from a few months to 16 years. The condition is characterized by recurrent attacks, often sudden in onset, of fatigue, cyanosis, pallor, increasing dyspnea and acceleration of pulse rate. There is a troublesome cough often followed by vomiting. At times there is blood in the sputum or vomitus. There may be abdominal pain. The temperature at the height of an attack may reach 103° F. Severe attacks can be very alarming. The patient is restless and often apprehensive. Each attack lasts two to three days. Toward the termination, the patient shows pallor and often jaundice. There is increased liver dulness and the spleen is palpable; there is often clubbing of the fingers. The attack begins with tachycardia, followed in twelve to twenty-four hours by dyspnea, pallor and fever, with or without jaundice. The clinical picture is one of congestive cardiac failure, especially of the lesser circulation. Recovery often is rapid but during intervals there is debility, undernutrition and dyspnea upon exertion. The diagnosis of hemolytic jaundice often is made. The true nature of the condition is unnoticed unless the chest is X-rayed. Between attacks, the pulmonary physical signs are insignificant; there may be a few scattered rhonchi. X-rays show mottled shadows which are most noticeable in the hilar areas. There is diffuse speckling throughout the lung fields. The abnormal shadows are accentuated during the attacks. There is often evidence of a partial lobar collapse. The shadows resemble those of miliary tuberculosis, except that, instead of dots, there are small, clear circular spaces surrounded by thickened opaque walls, giving a pumice-stone appearance. There is a good deal of similarity to the picture of sarcoidosis or Gaucher's disease. The cardiac shadow is enlarged to the right and the pulmonary conus is prominent. Examination of the blood shows a severe type of secondary hypochromic anemia with high reticulocytosis. Of the 17 previously reported cases, 5 were male and 10 female. The onset occurred in infancy or early childhood. There was no familial relationship among patients. All but one patient died with signs of left heart failure after several years of ill health. At autopsy, the main lesions are found in the lungs. Macroscopically, these are full and firm, of a dark reddish-brown color, with fine hemorrhages visable on the pleural surfaces. On section, there is pneumonic hepatization with a dark red-brown color of the cut surfaces. Enlargement of the tracheobronchial lymph nodes is conspicuous. The heart is enlarged, especially on the right side. Microscopically the alveoli in large areas are crowded with hemosiderin-laden phagocytes. The epithelial cells of the alveolar walls also contain hemosiderin; many are desquamated within

the alveoli, which also contain red cells and hemosiderin granules lying free. There is considerable thickening of the alveolar walls and interlobular and peribronchial tissues. Hemosiderin granules are contained within the interstitial tissues and also the nodes. The present series consists of 7 cases, 2 male and 5 female. The oldest was 13 years of age. No consanguinity was evident. Two patients have died. Of the 5 who are living, 3 are still subject to attacks. Tabulation of the clinical features shows hemoptysis or hematemesis in 6, positive chest X-ray findings in 7, palpable spleen in 3 and cardiac enlargement in 4 cases. Blood studies show hypochromia and increased reticulocytes; white blood and differential counts, platelets, clotting and bleeding times and red cell fragility all are normal. The anemia tends to subside in remissions, especially if the patient is given iron. Certain features such as reticulocytosis, nucleated red cells, slight jaundice, indirect van den Bergh reaction and slight increase in urinary urobilinogen suggest hemolytic anemia. But all these findings can be explained by the recurrent pulmonary hemorrhages with subsequent hemolysis. Histologically, even the youngest cases show evidence of capillary stasis and aggregation of heart failure cells and fresh red cells in the alveolar spaces. There is progressive thickening of the fibromuscular elements in the alveolar septa. Giant cells appear, which engulf the elastic fragments, and travel to the lymph nodes. The interalveolar capillaries become dilated and tortuous. Changes also appear in the small and medium-sized vessels. The bronchi are normal. The increase of reticulin, collagen and muscle and the decrease of elastic fibers results in a lack of distensibility of lungs, with consequent peripheral stasis in the capillary bed. This is followed by diapedesis and deposition of hemosiderin. Changes are slight at first. Gradually, larger portions of the lungs become involved. This explains the periodic course and increasing severity with eventual anoxemia. The hemosiderin deposits, fibrosis and collapse form the pathological basis for the radiological appearance. Pulmonary hemosiderosis had no relationship to generalized hemosiderosis and hemochromatosis. There is no disturbance in iron metabolism.—*Idiopathic Pulmonary Hemosiderosis, W. G. Wyllie, W. Sheldon, M. Bodian & A. Barlow, Quart. J. Med., January, 1948, 17: 25.*—(*A. G. Cohen*)

**Mediastinal Emphysema.**—From March 15, 1943 to March 15, 1947, at the University of Wisconsin, the author observed 6 students with spontaneous pneumothorax without recognized mediastinal emphysema, 7 with pneumomediastinum without recognized pneumothorax and 7 with both conditions. Spontaneous mediastinal emphysema occurs when air from a ruptured alveolus dissects along the perivascular sheaths to the mediastinum. Associated pneumothorax may be caused by rupture of the mediastinal air through the mediastinal pleura. It is probable that this mechanism operates in the majority of cases of spontaneous pneumothorax in otherwise healthy individuals; the initial pneumomediastinum may be transient or unrecognized. In mediastinal emphysema there is a characteristic sudden onset of pain, usually substernal and associated with change in position. The case histories of 14 cases of mediastinal emphysema are summarized. Six were aware of peculiar sounds over the precordium. A crunching sound synchronous with the heart beat is pathognomonic. Hyperresonance over the precordium was a constant finding in the author's series. In 3 patients, mediastinal air was demonstrated either by X-ray or fluoroscope. Electrocardiograms revealed no characteristic pattern. There was no significant change in leucocyte count or sedimentation rate. The occurrence of pneumothorax does not alter the course of mediastinal emphysema. Four of the 20 patients were tuberculin-positive. No student showed any lesions regarded as tuberculous in nature. Hospitalization of patients with spontaneous pneumothorax was advised only when seen early or when definite symptoms were present. Aspiration of air was carried out only for dyspnea or, in large collections, to hasten expansion of the collapsed lung. Prophylactic use of

sulfonamides or antibiotics is not recommended.—*Spontaneous Mediastinal Emphysema and Spontaneous Pneumothorax—A Report of 20 Cases, Helen A. Dickie, Ann. Int. Med., March, 1948, 28: 618.*—(*H. R. Nayer*)

**Bronchial Asthma.**—In view of the conflicting and incomplete statements in the literature concerning pulmonary ventilation, oxygen consumption and blood pressure values in bronchial asthma, this study was made to find out whether the results previously obtained by the author in experimentally induced asthma in guinea pigs could be verified in tests on asthmatic patients during and after the attack. The effect of the antiasthmatic drugs used in this investigation were studied on healthy persons, as well as on asthmatics during asthma-free periods and taken into account. It was found that, in accordance with the animal experiments, the pulmonary ventilation and oxygen consumption during severe attacks are lower than under normal conditions. Thus, an oxygen deficit occurs and this is made up by a considerable increase in the oxygen intake after the attack. In mild attacks, ventilation and oxygen intake are considerably greater than normal, the increment in the latter being as great as 100 per cent. In severe attacks, the systolic and diastolic blood pressures are considerably increased (the latter not constantly); in mild attacks, there is either insignificant or no increase. The altered gaseous tension seems to bring about changes in the intermediate metabolism probably affecting the condition of the patient. The author has previously demonstrated in guinea pigs that such insufficient metabolism causes a decrease in tissue reparation; this results from the break-down of co-zymase and co-phosphorylase.—*H. Colldahl, On the Pathophysiological and Clinical Aspects of the Crises of Asthma Bronchiale, I., The Pulmonary Ventilation, Oxygen Uptake and Blood Pressure, Act. Med. Scand., Sept., 1947, 128: 551.*—(*O. Pinner*)

**Bronchial Asthma.**—Temperature studies show that in severe attacks of bronchial asthma the temperature of the body falls, while in mild attacks a rise occurs. These changes are the same as previously found by the author in experimentally induced asthma in guinea pigs. However, in asthmatic patients it is difficult to decide what the influence of the concurrent bronchitis may be. Especially, retention of bronchial secretion may bring about temperature elevation. But the decline of the temperature during severe attacks cannot be due to infection, and the assumption that elevation in mild attacks is always a toxic manifestation of bronchitis is contradicted by the fact that in certain cases the temperature rises simultaneously with the transition of severe to mild asthmatic symptoms. The toxic influence of a bronchitis on the heat-regulating centers should be strongest at a time when the asthma attack is most severe and the sputum is tenacious and small in amount, but not when the attack and sputum retention are subsiding. Finally, the similarity of the temperature curves in experimental and in bronchial asthma argues against the theory of an infectious cause of the temperature rise. All this indicates rather that the asthmatic breathing as such has a decisive influence on the temperature. The mechanism involved, arising from the peculiarities of ventilation and oxygen consumption in mild and severe asthma, respectively, is analyzed in this paper. Asthma with an irregular course and with alternating mild and severe attacks shows an irregular temperature curve. A moderate rise in temperature of an asthmatic patient must, therefore, not be interpreted in all cases as due to infection.—*On the Pathological and Clinical Aspects of the Crises of Asthma Bronchiale, II, Effects of the Crises on the Body Temperature, Helge Colldahl, Act. Med. Scand., Oct., 1947, 129: 19.*—(*O. Pinner*)

**Tuberculin Sensitivity.**—The object of this study was to determine the relationship, if any, between the size of the tuberculin reaction and the presence and degree of tuberculous infection. Old Tuberculin in doses of 0.05 mg. and (if negative) 1.0 mg. were injected intracutaneously; the reactions were

interpreted in 48 to 72 hours. A total of 610 persons over the age of 15 were tested; this number included both healthy persons (contacts, etc.) and clinical cases. The size of the reaction was graded. Among nontuberculous individuals, the most intense reactions were noted in persons who were contacts with open cases. Milder reactions were found in both noncontacts or contacts with negative cases. The least intense reactions were seen in noncontacts. As the cases were followed, the strong reactions tended to grow milder. Increased sensitivity was found in cases with erythema nodosum and in primary tuberculosis, especially during the early phases. In clinical pulmonary tuberculosis, the reaction was greater in the exudative than in the productive type. It gradually decreased to a constant point. In healed cases, a new exposure often resulted in an increased reaction without the appearance of new lesions. In cavitary tuberculosis, the sensitivity was was low.—*Significance of Tuberculin Sensitivity, I. Bluhm, Tubercle, April, 1948, 29: 73.*—(*A. G. Cohen*)

**Chemotherapy of Tuberculosis.**—L-rhodinic acid and d-citronellic acid and the hydrogenated product of l-rhodinic acid, i.e., l-dihydro-rhodinic acid, were applied by mouth to 68 patients suffering from advanced pulmonary tuberculosis. Their course before and after the application was observed, as well as the fluctuation of tubercle bacilli in sputum. Data on 28 cases among the 68 patients are shown. The chief clinical phenomena which appeared on applying these fatty acids were: diminution or even disappearance of tubercle bacilli in sputum, regression of exudative lesions as shown by X-ray films, and sometimes disappearance of cavities, and decrease in sedimentation rate.—*Ueber die chemotherapeutische Anwendung von 1-Rhodinsäure, einem Bestandteil des Oels des Taiwanhinoki-Baums, und von einigen naheverwandten Fettsäuren bei tuberkulösen Patienten, S. Katsura, T. Nozoe & co-workers, J. Med. A. Formosa, 1941, 40: 1557.*—(*E. R. Long*)

**Chemotherapy of Tuberculosis.**—Investigations on the treatment of pulmonary tuberculosis with certain fatty acids were extended. Longer follow-up studies were carried out and, in addition to 1-rhodinic and d-citronellic acid, geranic acid and its hydrogenated products, dihydro-geranic acid (dl-dihydro-geranic acid) and tetrahydro-geranic acid, were tested. The results were qualitatively similar to those of the former report. Tubercle bacilli disappeared in 40 of 193 (20.7 per cent) cases. They disappeared more frequently in cases with comparatively small changes than in those with large changes, *e.g.* they disappeared in 21 out of 45 (46.7 per cent) cases without cavity images, in 29 out of 70 (41.4 per cent) cases with changes in localized fields, and in 15 out of 46 (32.6 per cent) cases with unilateral changes; while they disappeared in 19 out of 148 (12.8 per cent) cases with cavity images, in 11 out of 123 (8.9 per cent) cases with changes in total fields, and in 25 out of 147 (17.0 per cent) cases with bilateral changes. Although no parallel relation was recognized between the number of tubercle bacilli before treatment and their disappearance, yet their disappearance in 18 out of 112 (16.1 per cent) cases, which had corresponded to Gaffky VII-X, was notable. Cavity images disappeared in 18 out of 148 (12.2 per cent) cases, and infiltration shadows without cavity images were remarkably absorbed or disappeared in 27 out of 45 (60.0 per cent) cases. It is suggested that the side chains of the fatty acids might play an important rôle in the diminution of tubercle bacilli.—*Über die chemotherapeutische Anwendung von l-Rhodinsäure, einem Besandteil des Oels vom Taiwanhinoki-Baum, und von einigen anderen Fettsäuren naheverwandter abnormer Struktur bei tuberkulösen Patienten. (Zweite Mitteilung), S. Katsura, T. Nozoe & co-workers, Studia Medicinae Tropicalis, Supplementum I, 1944, 1: 352.*—(*E. R. Long*)

**Streptomycin and Promizole in Tuberculous Meningitis.**—Seven children with tuberculous meningitis were treated with streptomycin and promizole. Promizole was administered

orally at six or twelve hour intervals. The total daily dosage was 0.5 to 1.0 g. The daily dose was gradually increased until a blood level of 2 to 3 mg. per hundred cubic centimeters was obtained. The dosage of streptomycin was 0.5 to 1.0 g. in infants and small children, and 2 g. in older children. It was administered in divided doses at six hour intervals. In most cases 0.1 g. of streptomycin intrathecally on alternate days was well tolerated for about six weeks. Intrathecal therapy was then given only every third or fourth day amounting to a total of twenty-five to forty injections. No serious toxic effects of promizole were noted. One patient died while 6 are living three to eight months after treatment was instituted. The only neurologic sequelae are strabismus in one case and transient coarse tremors in another. The sugar content of the spinal fluid returned to normal in three to twelve weeks.—*Tuberculous Meningitis in Children, A Preliminary Report of its Treatment with Streptomycin and 'Promizole', Edith M. Lincon, T. W. Krimse & Estelle De Vito, J. A. M. A., February 28, 1948, 136: 593.—(H. Abeles)*

**Streptomycin in Tuberculous Meningitis.**—This article deals with the use of streptomycin in tuberculous meningitis as seen in children. Symptoms which should immediately make one suspect meningitis in tuberculin-positive children are headache, vomiting, sudden anorexia, fever (which is always present), and emaciation. Symptoms of longer standing, in addition to the above, will include personality changes and insomnia. Lumbar puncture should be a routine procedure which may have to be repeated. Choroidal tubercles and changes in the optic disk are frequently discovered. Forty-one of 90 cases acid-fast bacilli were found in the spinal fluid, and in 4 of 90 cases the Pirquet test was negative but the intradermal reaction was positive. Therefore, it is concluded that streptomycin should not be withheld if acid-fast bacilli are not recovered. Under streptomycin treatment, cases may terminate as follows: (*1*) deterioration whatever action is taken; (*2*) encouraging improvement but ultimate chronicity; (*3*) favorable progress and return to excellent health. In the last instance it cannot be known whether relapses will occur. At the beginning of this series, large doses of 100,000 to 200,000 units per kilogram per day were administered, along with intraspinal injections. With such large doses it becomes impossible to distinguish between symptoms and signs caused by the drug or by the disease. Large doses injected intraspinally always produce violent histological reactions, with lymphocyte counts ranging from 50 to 800 cells per cm. The dose of streptomycin in the latter half of the series was reduced to 100,000 units per kilogram per day parenterally and 50,000 to 100,000 units per day intraspinally during the first several days or week. This was again reduced to 50,000 units per kilo per day parenterally with no spinal injections. Under treatment, choroidal tubercles lose their original aspect and gradually heal or disappear, leaving residual pigment. The cell count in the spinal fluid returns to normal or subnormal, always below 10 and generally under 4. This occurred in 16 of the cases under observation. At the present time 46 children who have been undergoing treatment for from two to ten months appear to be normal. Treatment is stopped when there is an absence of all clinical meningeal symptoms, an ascending weight curve, complete apyrexia, normal sedimentation rate, a spinal lymphocyte count below 10 per cm., absence of tubercle bacilli in culture, and, if possible, a normal quantity of cerebrospinal albumin. A group of patients apparently cured of tuberculous meningitis, but still having symptoms and positive neurological signs, constitute a serious problem. It was decided to operate in 6 of these cases. In 5 of these, widespread leptomeningitis or pachymeningitis of the cisterna pontis was found; in the 6th there was a large meningeal tubercle about the chiasma. In 2 of these cases the results were remarkable. One patient, who was comatose and blind at the time of operation, is one who now gives the greatest hope of recovery.—*Streptomycin and Tuberculous*

*Meningitis in Children, Preliminary Note, R. D. St. Thieffry, E. Brissaud & H. Nouffard, Brit. M. J., December 6, 1947, 4535: 897.—(R. W. Clarke)*

**Pressure Breathing and Circulation.**—Simultaneous measurements were made during pressure breathing of the increase in volume of the lower leg, which was enclosed in a plethysmograph, and of the increase in weight of the caudal end of the body as indicated by the change in balance of a teeter-board on which the subject lay supine. From these results it may be calculated that an increase of pulmonary pressure of 30 cm. of water displaces 500 cc., or about half of the blood contained in the lungs; this represents about 8 to 10 per cent of the total blood volume. About 3 per cent of the total blood volume goes into the extremities and the remainder into the abdomen. In the standing position, there is less blood in the lungs, and the amount which can be displaced by pressure breathing is correspondingly less. By the use of a boot plethysmograph it is shown that the onset of pressure breathing causes an increase in foot volume due to passive inflation of the veins. If these passive changes are avoided by the previous inflation of a pneumatic cuff at 60 mm. Hg placed on the leg above the plethysmograph, then the onset of pressure breathing causes a decrease in the volume of the leg due to vasoconstriction. (Authors' Summary).—*Displacement of Blood from the Lungs by Pressure Breathing, W. O. Fenn, A. B. Otis, H. Rahn, L. E. Chadwick & A. H. Hegnauer, Am. J. Physiol., December, 1947, 151: 258.—(G. C. Leiner)*

**Pressure Breathing and Circulation.**—Positive pressure breathing produces vasoconstriction in the fingers and, therefore, a decrease in the finger pulse volume and a decrease in blood flow through the fingers.—*Effect of Pressure Breathing on Blood Flow through the Finger, W. O Fenn & L. E. Chadwick, Am. J. Physiol., December, 1947, 151: 270.—(G. C. Leiner)*

**BCG Vaccination.**—During 1924 to 1926, all new student nurses were given a Pirquet test immediately before assuming their duties among tuberculous patients. Very few clinical cases developed in the positive group, but many did in the negative group. In 1927, of 57 nonreactors, 45 consented to be vaccinated with BCG. During subsequent years, the same policy of offering BCG to nonreactors was followed. Each year, the morbidity in the vaccinated group was much less than in the nonvaccinated group and often as low as in the Priquet-positive group. The protective effect of BCG appeared to be durable.—*B.C.G. Vaccination of Nurses, J. Heimbeck, Tubercle, April, 1948, 29: 84.—(A. G. Cohen)*

**Apparatus for Airborne Infection.**—The author describes in detail an apparatus by means of which animals can be safely exposed to known numbers of droplet nuclei containing various infective agents. This is particularly suitable for quantitative natural airborne tuberculous infection. Only the fundamental principles of the device can be described here. A constant number of droplet nuclei of the infectious agent, engendered by atomizing a suspension of the agent with compressed air through a specially designed nozzle, is introduced into a chamber in which the animals are exposed, together with a constant flow of room air which is drawn into the chamber by draught action of a hot flame at the bottom of a specially constructed chimney which incinerates the outflowing infected air. The concentration of the infectious agent inhaled by the exposed animals is determined by the Wells air centrifuge. The tidal air of animals exposed can be measured during their sojourn in the apparatus. The entire system works under negative pressure and thus minimizes the hazard to the personnel. The instrument has been calibrated for the administration of minimal doses of tubercle bacilli to rabbits. It has been used for the study of the behaviour of inhaled particles in different states of suspension, as well as for the study of primary and reinfection tubercu

losis.—*On the Mechanics of Droplet Nuclei Infection. I. Apparatus for the Quantitative Study of Droplet Nuclei Infection in Animals, W. F. Wells, Am. J. Hyg., January, 1948, 47: 1.*—(*Max B. Lurie*)

**Quantitative Relation between Inhaled Tubercle Bacilli and the Resulting Tubercles.**—Particles containing bovine tubercle bacilli of such a size that they settled less than one-tenth of a foot per minute in still air were introduced into the apparatus described in the preceding paper. If such particles containing single bacilli are inhaled by rabbits, it is claimed that each organism will give rise to a tubercle in the lung. If the particles are coarse so that they settle one foot per minute, only one in ten such particles will induce a tubercle in the lungs of rabbits; the remainedr are swallowed. As has been proven many times before, it is shown here again that a first infection protects from a subsequent inhalation reinfection. The theoretical basis for the above conclusions must be pursued in the paper proper. *On the Mechanics of Droplet Nuclei Infection. II. Quantitative Experimental Air-Borne Tuberculosis in Rabbits, W. F. Wells, H. L. Ratcliffe & C. Crumb, Am. J. Hyg., January, 1948, 47: 11.*—(*Max B. Lurie*)

**Tomography of the Spine in Tuberculous Disease.**—Tuberculosis of the spine is first noticeable in the X-ray as the diminution of the space between the vertebral bodies with decalcification of the adjacent portions of bone. As caries progresses some collapse occurs and several vertebral bodies may be involved. Ordinary anteroposterior and lateral X-rays are useful in finding the extent and progress of the disease. Tomography is of special value, however, in overcoming difficulty in such areas as the first and second cervical vertebrae, the cervical dorsal region, and the lumbosacral area, where overlapping structures may interfere in delineation of bony disease. In the region of the first and second cervical vertebrae, a good view of the diseased area may be obtained from an ordinary lateral film, but in the anteroposterior X-ray, the area is usually obscured by the lower jaw. In the cervical dorsal region, tuberculous disease may be delineated with plain films, but tomography may be very helpful in more accurate definition. Tuberculous disease of the sacrum is rare except in conjunction with disease of the sacroiliac joints. Tomograms may be of value in special cases. A brief description of the radiographic technique is given.—*Tomography of the Spine in Tuberculous Disease, S. G. Wood & M. C. Wilkinson, Brit. J. Radiol., October, 1947, 20: 418.*—(*L. Hyde*)

**Lung Abscess.**—During a period of ten years, pulmonary resection for lung abscess was performed in 37 cases. This number includes only those which appeared to be abscess cases from the very beginning, and not secondary to other conditions. The patients had all received medical treatment, often including bronchoscopic aspiration and the use of sulfonamide drugs and penicillin. The condition developed in the course of respiratory infection in 20 cases, following inhalation anesthesia in 11, complicating a foreign body in 5, and after a chest injury in one. Seven patients had had previous surgical therapy consisting of artificial pneumothorax in 2, rib resection and drainage in 3, drainage of empyema in one and drainage and thoracoplasty in one. The average time from diagnosis to resection was eighteen months. The operation consisted of segmental resection in 2, lobectomy in 16 and bilobectomy in 3 cases. Of these 21 cases, there was one operative death. Complications occurred in 3 cases, all of which responded to treatment. Pneumonectomy was performed in 16 cases. There were 6 operative deaths. In addition, 3 patients died of brain abscess within five months. Additional, nonfatal complications were noted in 3 cases. The recent trend in the treatment of chronic lung abscess is away from drainage and toward resection. The stated indications for resection are: (*1*) persistent symptoms due to pathological changes secondary to open drainage; (*2*) multiple or multilocular

abscesses; (*3*) abscesses associated with secondary changes, consisting of fibrosis, bronchiectasis, bronchostenosis and atelectasis; (*4*) abscesses so located anatomically as to be inaccessible to adequate drainage; (*5*) abscesses in which the diagnosis of malignancy is entertained; (*6*) abscesses associated from the onset with excessive bleeding; (*7*) abscesses in children; (*8*) abscesses secondary to foreign bodies not removable by bronchoscopy. —*Pulmonary Resection for Abscess of The Lung, R. P. Glover & O. T. Clagett, Surg., Gynec. & Obst., April, 1948, 86: 385.*—(*A. G. Cohen*)

**Cough as a Symptom.**—Cough is dependent upon a local stimulus which originates nerve reflexes through the medulla. The act of coughing can be divided into three phases: (*1*) the inspiratory phase, followed by (*2*) the compressive phase which depends upon the closure of the glottis and an increase in the intrapulmonary pressure immediately preceding expiration, and (*3*) the expiratory phase, when the vocal cords and ventricular bands are quickly separated and air is forced out with the production of characteristic cough sounds. Purpose of cough is the removal of mucus, inflammatory exudate, and any other material in the air passages. The rôle played by bronchial movements during respiration is extremely important in the removal of secretions from the air passages. Deep inspiration increases the motion of the bronchial walls and this tends to move bronchial secretions. The degree of mobility is practically negligible during quiet respiration, such as is observed in narcosis, acute pleurisy and injuries to the chest. Ciliary function is also important in the elimination of secretions from the air passages. It is more effective in the presence of thin than of thick secretions. Cough is a symptom and the cause must be determined. It may be caused by common respiratory tract disease, smoking, exposure to dust and fumes, extra-respiratory factors ("ear cough" or "reflex aural cough"), diseases of the nose and nasal accessory sinuses, nasal or pharyngeal obstruction, aspiration of food or fluid, and allergy. Cough of functional origin occurs, but one must be careful to exclude all possible organic causes before arriving at this diagnosis. To determine the exact cause of cough, careful history and physical examination are necessary. Examination of the ear, nose and throat, and chest, plus chest X-rays, are usually indicated. Bronchography is indicated if there is any suspicion of increased bronchopulmonary markings suggesting bronchiectasis. In the presence of a history of allergy, appropriate tests must be performed. Treatment must be directed at the cause. Cough is necessary to rid the tracheal bronchial tree of excessive secretions, for example, from pulmonary abscess or bronchiectasis, and in these conditions narcotics should be used sparingly if at all. On the other hand in carcinoma cough is quite purposeless, and therefore symptomatic therapy and suppression may be indicated. When cough is inadequate due to thick tenacious secretions, so-called stimulating expectorants are recommended. Inhalations of carbon dioxide and oxygen increase the quantity of sputum and have been highly recommended.—*Cough as a Symptom, L. H. Clerf, M. Clin. North America, November, 1947, 31: 1393.*—(*L. Hyde*)

**Cryptococcosis.**—The case history of a girl whose disease started in 1937, and who was first reported in 1941, is reviewed. In 1938, *Cryptococcus hominis* was found in the material drained from a chest sinus and in the spinal fluid. Since then the patient has had several courses of sulfadiazine treatment; the lesions in the chest have improved while the changes in the central nervous system have shown slow progression. The patient is alive nine years after the onset of symptoms. On the basis of experiments on rats infected with cultures of *Cryptococcus hominis*, the following conclusions are made: "Animals previously infected with the cryptococcus and clinically appearing well gave no sign of having developed antibody protection against that organism. Animals treated with sulfadiazine

had their life expectancy increased from 11.4 to 28.9 days. Animals treated with penicillin had their life expectancy increased from 11.4 to 22.6 days. No advantage accrued when penicillin and sulfadiazine were used in combination. Animals treated with streptomycin had their life expectancy increased from 11.4 to 28.0 days."—*Cryptococcosis. Report of a Case and Experimental Studies, E. B. Reilly & E. L. Artman, Arch. Int. Med., January, 1948, 81: 1.*—(*G. C. Leiner*)

**Pericarditis following Upper Respiratory Infection.**—A 52-year-old man developed tonsillitis with fever. Within three weeks pericarditis with effusion had occurred. Culture of the fluid showed hemolytic streptococcus. Repeated cultures from an accompanying pleural effusion were sterile. The patient recovered completely after two courses of penicillin treatment.—*Pericardial Effusion due to Hemolytic Streptococcus following an Acute Upper Respiratory Infection; Associated Pleural Effusion; Report of Case and Review of Literature, S. H. Rinzler & S. Leibowitz, Am. Heart J., March, 1948, 35: 490.*—(*G. C. Leiner*)

**Mediastinitis.**—Two cases of mediastinitis in children are reported. Both followed the ingestion of baby food from containers from which glass had been chipped when the containers were opened. One case was proved to be due to the ingestion of the glass fragment, while the other was presumably due to the same type of fragment (Author's Summary).—*Mediastinitis due to Ingestion of Glass, J. D. Steele, J. A. M. A., February 21, 1948, 136: 554.*—(*H. Abeles*)

**Mediastinotomy.**—A case of spontaneous mediastinal emphysema requiring mediastinotomy is presented. The clinical picture, diagnosis and management are discussed (Authors' Summary).—*Mediastinotomy in Spontaneous Mediastinal Emphysema, J. R. Karns & E. O. Daue, Jr., J. A. M. A., February 28, 1948, 136: 622.*—(*H. Abeles*)

**Silicosis.**—Every new person employed in the Calumet industrial area was given a physical and X-ray examination. The films were graded on the basis of the extent of lung markings plus the size of lymph nodes. There was a fairly close correlation between the extent of these changes and the length of time a person had worked in this area. These changes were not disqualifying. Apical scarring or other evidence of tuberculosis was disqualifying. Repeat X-rays were taken every year. The earliest changes resulting from dust were seen in six years, with an average of fifteen years. The changes consisted of enlargement and thickening of the hilar nodes. These were termed potential primary silicosis, and were not associated with symptoms. If the changes were progressive in workers under 45 years of age, removal from exposure to silica was advised. If over 45, the workers were allowed to continue unless there was evidence of pulmonary infection. A definite diagnosis of silicosis was not made unless there was nodule formation in the parenchyma. The onset of tuberculosis was very insidious. Aluminum therapy was provided on a voluntary basis; treatment was not given if there was evidence of tuberculosis. In the prophylactic group 30 to 40 treatments were given anually while in the therapeutic group, 40 treatments were given. Treatment consisted of the inhalation of aluminum dust or oxide through the mouth. The first treatment lasted two to three minutes. The time was gradually increased to ten minutes by the eighth treatment and so continued. There were no toxic reactions. It is believed that the progression of the silicosis was checked markedly by aluminum therapy and that the incidence of secondary tuberculosis was much less. The authors are not certain of the benefits of prophylaxis, but believe them to be good.—*Silicosis Study and Management in the Calumet Industrial Area, C. W. Rauschenbach, D. R. Johns, J. F. Larabee, L. M. Hammar & B. F. Poracky, Indust. Med., January, 1948, 17: 1.*—(*A. G. Cohen*)

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